

CORRESPONDENCE

What other treasures could be hidden in conference papers?

SIR — In the obituary of Anatol Zhabotinsky (*Nature* **455**, 1053; 2008), Irving Epstein mentions Boris Belousov, with whom Zhabotinsky shared the Lenin Prize in 1980 for their contributions to the Belousov–Zhabotinsky oscillatory chemical reaction system.

Epstein says “Belousov tried to publish his results in peer-reviewed journals, but eventually gave up after referees and editors insisted that such behaviour contradicted the Second Law of Thermodynamics. He instead published a one-page description of his observations in an obscure conference proceedings on radiation medicine.” That paper¹, ‘A periodic reaction and its mechanism’, gained little attention at the time.

Papers published in symposium proceedings do not usually merit citation, because they are not peer-reviewed. They receive little recognition. Very few are even indexed in the main journal databases — one notable exception being PubMed’s listing of the annual Cold Spring Harbor Symposium on Quantitative Biology.

However, other ‘hidden’ conference papers have also subsequently provoked acclaim. The pioneering work of physicist Abdus Salam and chemist Koichi Tanaka aroused little interest when it was first published in this way^{2,3}. Fortunately, these findings were later recognized for their originality and importance: Salam went on to win the 1979 Nobel Prize in Physics, and Tanaka was awarded the 2002 Nobel Prize in Chemistry.

Min-Liang Wong Department of Veterinary Medicine, National Chung-Hsing University, Taichung 402, Taiwan
e-mail: mlwong@dragon.nchu.edu.tw

1. Belousov, B. P. *Compil. Abstr. Radiat. Med.* **147**, 145 (1959).
2. Salam, A. in *Elementary Particle Theory, Proceedings of the Nobel Symposium held in*

1968 at Lerum, Sweden (ed. Svartholm, N.) 367–377 (Almqvist & Wiksell, 1968).

3. Tanaka, K. *et al.* in *Proceedings of the Second Japan–China Joint Symposium on Mass Spectrometry* (eds Matsuda, H. and Liang X. T.) 185–188 (Bando, 1987).

Public opinion and the ethics of primate brain research

SIR — Several issues in your News story ‘German authority halts primate work’ (*Nature* **455**, 1159; 2008) call for clarification. Freedom of research is written into German basic law, but so is animal protection. This reflects concerns in society at large about the ethics of subjecting animals to pain and distress in research, as well as in farming and for entertainment. Even a prominent scientist such as Andreas Kreiter must justify his use of animals.

Although Kreiter refers to the ethical judgement by Bremen’s senate of health as “purely arbitrary”, it is backed by a political majority in the Bremen Senate as well as by the majority of Bremen’s citizens, as confirmed in petitions and opinion polls.

You say that “the ruling ignores a positive judgement rendered last year by an expert commission comprising scientists and representatives of animal-welfare organizations”. But the commission restricted itself to assessing the scientific merits of Kreiter’s research, not the ethical issues — thereby failing in part of its mandate, which expressly included ethical issues.

Also, there was only a single animal-welfare specialist among the five members of this commission; the remainder were scientists who conduct brain research in primates or breed them for research. The previous year, one of them had himself been denied permission, on ethical grounds, to conduct invasive brain research. Moreover, you imply that the animal-welfare specialist also approved the monkey experiments. Nothing could be further from the truth.

This is not the first time that the expert group’s judgement has been misrepresented to the media by scientists and university officials. Again and again the German Animal Welfare Federation has been forced to try and correct the mistaken impression that experts in science, ethics and animal welfare unanimously endorsed Kreiter’s project.

Kreiter’s failure to explain satisfactorily to the public exactly what he is doing is seriously undermining his credibility, and that of scientists in general. Insisting that the ethical concerns are unreasonable and that the constitutional mandate of animal protection is an undemocratic assault on academic liberty deepens the antagonism between town and gown.

You quote Stefan Treue as saying he “just can’t see why what’s perfectly fine in one place should be unethical in another”. In fact, monkey-brain research much like Kreiter’s in the level of suffering it causes has been prohibited in Munich, Berlin and Zurich.

Kreiter’s centre for primate research in its present form is now in jeopardy. He should face the fact that ethical standards have evolved since he started this work and that he has lost touch with the majority of his fellow citizens.

Ulrike Gross German Animal Welfare Federation, Animal Welfare Academy, Spechtstraße 1, 85579 Neubiberg, Germany
e-mail: ulrike.gross@tierschutzakademie.de

Marker metabolites can be therapeutic targets as well

SIR — Your News & Views Q&A article ‘Systems biology: metabonomics’ (*Nature* **455**, 1054–1056; 2008) highlights the importance of metabonomics in the identification of metabolites associated with disease — for example, as biological markers for disease state and susceptibility, and for monitoring

response to treatment. However, metabonomics can also be useful for determining the therapeutic potential of metabolites whose levels are altered in a particular disease state.

If changing concentrations of a specific metabolite can be linked to the genesis or progression of a disease, then there may be a therapeutic advantage in restoring these to normal values. This strategy has been successful, or at least promising, in many cases. For example, several anticancer treatments exploit the antiproliferative action of ceramide, the concentration of which decreases in certain cancer types (C. P. Reynolds *et al.* *Cancer Lett.* **206**, 169–180; 2004). Also, increasing the concentrations of S-nitrosothiol metabolites in the airway-lining fluid, which are lowered in patients with asthma, seems to have a protective effect in animal models (L. G. Que *et al.* *Science* **308**, 1618–1621; 2005).

Metabolites have a variety of cellular functions, including acting as direct regulators of gene expression, so it is not surprising that they can also function as effectors of molecular events that contribute to disease. Those positively associated with disease causation may be rarer than those that simply result from a disease.

The human metabolome comprises thousands of endogenous molecules, many of whose functions are unknown. We believe that the concept of disease-associated metabolites as potential therapeutic agents is underexploited, in comparison with their widespread use as biological markers.

Adrian K. Arakaki, **Jeffrey Skolnick** Center for the Study of Systems Biology, Georgia Institute of Technology, 250 14th Street NW, Atlanta, Georgia 30318, USA
e-mail: adrian.arakaki@gatech.edu
John F. McDonald School of Biology, Georgia Institute of Technology, 310 Ferst Drive, Atlanta, Georgia 30332, USA

Contributions may be submitted to correspondence@nature.com.